34. ZUR DIFFERENTIALDIAGNOSE DER MULTIPLEN SKLEROSE (Differential Diagnosis of Multiple Sclerosis). D. Gerhardt (Deutsche Zeitschrift für Nervenheilkunde, Vol. 15, 1899, p. 458).

Gerhardt reports a case in which he observed intention tremor, spasticity of the limbs, scanning speech, apoplectic attacks—in short, the clinical picture of disseminated sclerosis without nystagmus. The intelligence began to fail, dementia became pronounced, and muscular twitchings and grinding of the teeth were noticed. The necropsy showed macroscopically increased consistency of the brain and spinal cord, which was believed to be due to diffuse sclerosis, but microscopically this sclerosis was observed only in the cord. The case bore some resemblance also to one of general paralysis. Atrophy of the cerebrum, especially of the frontal lobes, slight chronic leptomeningitis, accumulations of cells and pigment in the vascular sheaths, destruction of tangential fibers—these were very like the findings of general paralysis, but there was no ependymitis granulosa. The dementia was different from that of general paralysis; it increased with the decrease of the apoplectic attacks, was not associated with ideas of grandeur, and was like the dementia of cerebral hemorrhage or softening. The mental disturbance occurs early in paretic dementia, but in Gerhardt's case it came late in the disease, and after the symptoms of multiple sclerosis were seen. Diffuse sclerosis should be thought of when the symptoms of multiple sclerosis are present without nystagmus, when mental failure becomes more and more prominent, when the scanning speech becomes more like that of general paralysis, and when muscular tremor and grinding of the teeth occur. This diffuse sclerosis cannot be separated clinically or histologically from Strümpell's pseudo-sclerosis.

Small multiple foci of softening as well as diffuse sclerosis can cause the clinical appearances of disseminated sclerosis, and a case of the kind is reported by Gerhardt. Usually multiple foci of softening cause bulbar symptoms, and the disease is more common later in life, while disseminated sclerosis is more common in early life. SPILLER.

35. LES DIFFÉRENTES FORMES DE PARAPLÉGIE DUES À LA COMPRESSION DE LA MOELLE EPINIÈRE ET LEUR PHYSIOLOGIE PATHOLOGIQUE (The General Symptoms of Paraplegia Due to Compression of the Spinal Cord, and its Pathological Physiology). A. van Gehuchten (La Presse médicale, 1899, p. 218).

In this exceedingly valuable article, van Gehuchten undertakes to analyze and explain the various symptoms produced by complete destruction of a transverse segment of the cord. He divides a typical case of paraplegia into four degrees. In the first there is spasmodic paraplegia, exaggeration of the reflexes, but no sensory disturbances. In the second, flaccid paraplegia, loss of reflexes, but still no sensory disturbances. In the third, the motor symptoms are the same as those in the second degree, but there is the syringomyelic dissociation of sensation; and in the fourth, the motor symptoms are still unchanged, but there is complete anesthesia. Between these types there are, however, an infinite number of intermediate stages. Naturally, all the four forms usually occur successively in the same patient, providing the cause of the lesion is persistent and progressive. Therefore, van Gehuchten has prepared a second and longer table in which he includes the most characteristic of the transitional forms. These, however, are essentially characterized by partial instead of complete sensory disturbances. In a brief review of the anatomy of the tracts of the cord, he includes among them the motor fibers, the antero-lateral pyramidal tracts, the cerebello-spinal tracts, uniting the cerebellum with the anterior cornua of the cord and motor fibers belonging to the posterior longitudinal fascicle that arises from a group of cells just beneath the oculo motor nucleus. He also believes that descending fibers pass into the spinal cord from the nucleus rubrum and the nucleus of Flechsig. Of all these fibers the most important are the cortico-spinal group, and in the first stage of compression they alone are involved and give rise to the syndrome of spasmodic paraplegia. As the pressure increases, all the descending fibers become involved, paraplegia becomes flaccid, and the reflexes are lost. Further compression produced alterations in sensation, involving first the fibers conveying pain and heat sensation which pass up through the gray matter of the cord, and ultimately the fibers of muscle sense which occupy the columns of Goll and Burdach. Sometimes there is isolated disturbance of pain sensation while heat sensation remains normal, or the area of disturbance of pain sensation is more disturbed than the area of heat sensation. Van Gehuchten questions whether the same fibers convey both forms. It is curious that although the fibers of the posterior columns in the cerebellar tract are most resistant to compression, they are most susceptible to syphilitic disease. There are many exceptions to these general rules and several cases are quoted, mostly from the literature, in which either the sensory disturbances occurred first, or there was flaccid paralysis with exaggeration of the reflexes. In a few rare cases there are motor disturbances with exaggeration of one or other forms of sensation; that is to say, some form of hyperesthesia. This is probably caused by the zone of hyperesthesia described in Brown-Sequard's paralysis. This hyperesthesia has been explained by Martinotti, as a result of the lesion of a narrow band of fibers that is situated in the angle between the anterior and posterior cornua. In all cases of total transverse destruction of the cord, hyperesthesia is absent. SAILER.

36. DES NÉVRALGIES ET TICS DE LA FACE CONSIDÉRÉS DANS LEURS RAPPORTS AVEC UN ÉTAT PATHOLOGIQUE DES VOIES LACRYMALES (The Neuralgias and Tics of the Face Considered in Their Relation with a Pathologic Condition of the Lachrymal Ducts). P. Bettremieux (Archives provinciales de médecine, Vol. 1, No. 10, October, 1899, p. 703).

The writer believes that there is a pathogenic relation between the neuralgias and tics of the face and diseased conditions of the lachrymo-nasal mucous membrane. He reports nine cases, four of neuralgia, three of tic convulsif, and two of tic douloureux, in which the results of his treatment tend to uphold his opinion. Of these nine cases, a marked improvement, or cure, was obtained in six, no result was obtained in one, and two were lost sight of. In nearly all of these cases there was increased lachrymation on the affected side, which had not been noticed by the patient. The treatment consisted of either simple catheterization of the lachrymal duct, catheterization with injections of nitrate of silver solution or some other antiseptic, or the passage of sounds in the duct followed by injections. There was almost immediate relief from pain in those cases in which it had been present after one treatment, and convulsive movements were diminished in severity. It seemed in some cases to be necessary to keep up the treatment for some time, or the trouble would return. Those cases which were not cured enjoyed a longer respite between the attacks, which also were not so severe as formerly. Bettremieux concludes that the lachrymo-nasal mucous membrane, if diseased, may be the seat of neuralgias and tics of the face, and that this lesion may be superficial,